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? ds

Set     Items   Description
S1      12987   EHEC OR ENTEROHAEMORRHAGIC OR ENTEROHEMORRHAGIC
S2          1   S1 AND TYPEIII
S3        734   S1 AND TYPE(W)III
S4         91   S3 AND PY<2001
S5         23   RD S4  (unique items)
S6         71   S1 AND CULTURE AND SUPERNATANT
S7         26   RD S6  (unique items)
S8          6   AU='FINLAY, BRETT' OR AU='FINLAY, BRETT B.'
S9        102   E1-E4
S10       107   S8 OR S9
S11         2   S1 AND S10
S12         2   RD S11  (unique items)
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7/7/17 (Item 2 from file: 50)
DIALOG(R)File 50:CAB Abstracts


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Culture negative cytotoxin positive stools in community acquired diarrhoea.

Burke, D.; Al Jumaili, B. J.; Al Mardini, H.; Record, C. O.
(C.O. Record) Royal Victoria Infirmary, Newcastle upon Tyne, NE1 4LP, England.

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Enterohaemorrhagic Escherichia coli (EHEC ) are being increasingly recognized as causative organisms of diarrhoea as well as other types of intestinal infections. However, apart from strains of ***EHEC*** belonging to serogroup O157 diagnosis is usually difficult. In addition cultures of Esch. coli often rapidly lose their ability to produce the characteristic Verocytotoxins (VT) on subculture making the identification of such ***EHEC*** by cultural methods also difficult. In this study in Newcastle upon Tyne (England) the authors examined stool specimens from patients aged over 10 years for the presence of VTs directly as well as performing the usual cultural methods to detect enteropathogens. A portion of the stool sample was emulsified in phosphate-buffered saline (PBS; 1 in 4) and then 5 ml of brain-heart infusion (BHI) broth inoculated with 1 ml of the PBS-stool suspension. After overnight incubation at 37 (deg)C this culture was added to 35 ml BHI broth in a 250 ml conical flask, after 1 h at 37 (deg)C mitomycin C was added to give a concentration of 1 microg/ml and the culture was incubated for a further 6 h at 37 (deg)C. The ***supernatant*** layer obtained from this culture on centrifugation and filtration through 0.22 microm millipore filter was tested for cytotoxicity against HeLa cells and in some cases against Vero cells. Of the 175 ***culture*** -negative samples that were studied, of which 162 were from suspected cases of infective diarrhoea and 13 from contacts of patients with diarrhoea, 51 (29.1%) showed a cytotoxic effect, of which 10 (5.7%) showed neutralization with antibody to VT1, 33 (18.9%) by antibody to VT2 and 8 (4.6%) were neutralized by neither. None of the samples showed neutralization by Clostridium sordelli antitoxin. None of the 25 stool specimens from healthy control individuals demonstrated the presence of any cytotoxins. These results indicate that there must be many ***EHEC*** present in the community causing diarrhoea.

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Effect of environmental conditions on proteins secreted by ***enterohemorrhagic*** Escherichia coli O26:H11.
AUTHOR: Yoh Myonsun (Reprint); Bi Zhenqing; Matsuyama Junko; Nagayama Kenichi; Honda Takeshi
AUTHOR ADDRESS: Research Center for Emerging Infectious Diseases, Research Institute for Microbial Diseases, Osaka University, 3-1 Yamadaoka, Suita, Osaka, 565-0871, Japan**Japan
AUTHOR E-MAIL ADDRESS: myon@biken.osaka-u.ac.jp
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ABSTRACT: Infections due to Shiga toxin-producing Escherichia coli (STEC) are responsible for severe diarrheal diseases in humans, and these bacteria have recently emerged as a leading cause of renal failure and encephalitis in children and the aged. In this study, we examined the environment-dependent production of proteins secreted from a strain of STEC O26:H11 by trichloroacetic acid precipitation, SDS-PAGE, Western blotting and N-terminal amino acid sequence analysis. Growth of bacteria in essential minimum medium (M9) led to the detection of secreted proteins of 104, 80, 40, 37 and 25 kDa (P104, P80, P40, P37 and P25, respectively). When grown in serum-free MEM, only P104, P40, P37 and P25 were observed in ***supernatant*** fluids. Growth of the bacteria in Luria-Bertani broth (LB) enhanced the expression of P104, but the productions of the other proteins were remarkably reduced. CO2 increased the secretion of P80 and P37, but reduced the production of P104. N-terminal amino acid sequencing revealed that P104 was EspP of STEC, which was homologous to EspC of enteropathogenic Escherichia coli (EPEC), and both proteins belong to a subclass of the IgA protease family. P80, which was identified as EspE of STEC, was homologous to Tir of EPEC. P40, P37 and P25 were found to be highly homologous to the similarly sized EspD, EspB and EspA proteins, previously detected in culture supernatants of EPEC. Those proteins are thought to be STEC virulence factors. Sera were obtained from two patients, one with colitis and another with hemolytic uremic syndrome (HUS), caused by STEC O157:H7, to study immune response to secreted proteins. Our results suggested that